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PLATE I.

ARTHUR TUDOR EDWARDS.

[*Frontispiece.*

BRITISH JOURNAL OF
TUBERCULOSIS
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IN MEMORIAM

ARTHUR TUDOR EDWARDS

ARTHUR TUDOR EDWARDS died on August 25 at the early age of fifty-six, after a life of full activity which undoubtedly contributed to his early demise.

He was born in Swansea in 1890, the son of William Edwards, J.P. He was educated at Mill Hill, and from there went on to St. John's College, Cambridge. His clinical studies were carried out at Middlesex Hospital.

Very early in his medical career his mind was set on surgery, and after doing resident appointments he became Surgical Registrar at the Middlesex. During the 1914-18 war he served as a Surgical Specialist with the rank of Major in the 16th Casualty Clearing Station, and was at that time recognised by his associates as a surgeon of no mean ability.

After the war in 1920 he was appointed Assistant Surgeon to Westminster Hospital, where at first his chief interest lay in urological and abdominal surgery. At this early stage in his career he impressed his co-workers with his technique, his unremitting care and attention to detail, and his courageous approach to difficult cases and awkward situations. Here at Westminster and at Queen Mary's Hospital, Roehampton, he had a considerable experience of general surgery, the value of which he never underestimated, for he always insisted that before any special branch of surgery was embarked upon, a sound training in general surgery was essential.

In 1921 he was appointed Surgeon to Brompton Hospital, and it was at this point that his real life work commenced. All the qualities with which he was possessed were unremittingly applied to the unexplored field of thoracic surgery. From the early beginnings when the natural and probably justifiable conservatism of that time made his work an uphill fight, by well-conceived and carefully planned interventions he established the value of thoracic surgery so well that today it is as much an independent branch as abdominal surgery. In 1929 he carried out the first dissection lobectomy to be performed in this country. The first pneumonectomy to be done lies to his credit. These, however, were but incidents in the whole fabric—it was his drive and example which had the greatest influence in the field in which he was undoubtedly one of the pioneers. His theatre at Brompton was a meeting place for all those interested in thoracic surgery, both at home and abroad, and very soon his reputation was internationally established in this field.

During this period his energy was terrific. At prolonged operating sessions he was apparently the least tired, and the last case had the same care as the first. Perhaps the most remarkable characteristic was that during these strenuous times he never lost his control of the situation; he rarely ever showed the least sign of irritability and was never known to lose his temper with an assistant, however unhelpful he might be. Carelessness or inattention to detail, however, he could not tolerate, and any attempt to mislead him in the attempt to cover such a lapse was severely dealt with, fairly, coldly and judiciously, never, however, before others but always *à deux*. Good service, on the other hand, always drew from him his approbation, often unexpressed in words, and in the rather shy, offhand manner so characteristic of him. He was fundamentally shy, and found the outward expression of his deepest feelings very difficult, a trait which was appreciated, unfortunately, only by the few who were intimate with him. Failure to appreciate this led many to think that he was cold, distant and difficult to approach. Once the crust of his shyness and reserve was broken, his essential kindliness and loyalty were easy to see.

In 1939 he had his first serious illness. In many this would have been sufficient cause to lead to a retirement from active life. Such a course was impossible for one so dynamic. Despite several recurrences, he carried on a full and active life, facing with the utmost courage the inevitability of an early death. During this time, as consultant adviser to the E.M.S., he organised the thoracic surgical service for the whole country for the reception of chest injuries. Due to his foresight, these centres were used not only for traumatic cases, but also for those with chest disease. The value of this is incalculable, for in this way a thoracic service was put at the disposal of large sections of the community hitherto unprovided for, leading to much greater demands on the service than had been thought possible.

Added to this and his other activities, he acted as Civilian Consultant to the R.A.F., opening a centre for this purpose at the King Edward VII Sanatorium, Midhurst, where he was Consulting Surgeon. He was also Consultant to the Army at Millbank, and through the many committees of which he was a member co-ordinated the civilian and Services thoracic surgical work.

He was a past president of the Society of Thoracic Surgeons of Great Britain and Ireland, and the first president of the Association for the Study of Diseases of the Chest, in the foundation of both of which he played a prominent part.

His pioneer work in his own particular field received ample recognition from abroad. He received the Honorary Fellowship of the American Association of Thoracic Surgeons, the M.D. Honoris Causa of the University of Grenoble, and just before his death the M.D. Hon. Caus. of the University of Oslo, which, owing to illness, he was unable to receive in person.

Tudor Edwards' death has robbed the profession of a great pioneer, a fine surgeon, and a true and loyal friend to all those who were associated with him.

C. P. T.

GENERAL ARTICLES

BRONCHO-PULMONARY ABNORMALITIES

By A. F. FOSTER-CARTER
From the Brompton Hospital Sanatorium, Frimley

Introduction

Abnormalities of the lung and bronchial tree are not only being recognised more often, as the study of thoracic disease advances, but they are also assuming a new importance because of their bearing upon diagnosis and treatment. The fresh material supplied by thoracic surgery, bronchoscopy and bronchography is also bringing to light new peculiarities hitherto unsuspected. In the past, differences in nomenclature and the employment of loose terms with ill-defined meanings have tended to confuse investigators, and there is a real need for some classification of pulmonary abnormalities which will sort out the known varieties and yet be elastic enough to allow for future additions.

It is not intended to attempt a comprehensive account of all the known aberrations of the lung in this short paper, but rather to describe some of the more common ones, showing their relationship one to another and their clinical significance. The work is based on examples which have been encountered in the course of routine clinical studies and while investigating the anatomy of the bronchi; they have only been described in detail when they supply some new fact or throw fresh light upon an old problem.

Table 1.—Classification of Broncho-Pulmonary Abnormalities

I. *Abnormalities of Lobation:*
- *a.* Additional fissures.
- *b.* Deficient fissures.
- *c.* Fissures due to displaced structures:
  1. Lobe of the azygos vein.
  2. Sequestration of lung due to an abnormal artery.
  3. Fissure for subclavian artery.

II. *Abnormalities of the Bronchi:*
- *a.* Supernumerary bronchi.
- *b.* Displaced bronchi.
- *c.* Congenital cystic disease.

III. *Abnormalities of the Parenchyma:*
- *a.* Accessory lungs.
- *b.* Agenesis of the lung.
- *c.* Hypoplasia of the lung.

It has been found convenient to arrange the common pulmonary abnormalities in three groups: (1) Abnormalities of lobation, (2) abnormalities of the bronchi, and (3) abnormalities of the parenchyma. A provisional classification under these headings is offered in Table 1. When considering any

such classification it must be remembered that individual cases may show anomalies belonging to more than one group, and that, in a variable structure like the lung, there is bound to be a considerable no-man's-land between the minor variation and the major abnormality. Only comparatively wide deviations from the common pattern will be considered here.

I. Abnormalities of Lobation

(*a*) *Additional Fissures*

The most common type of abnormal lung is one which has more or less than the usual number of lobes, owing to additional or poorly developed fissures. Such cases have often been described in the past, and in some instances names have been given to the sequestered areas of lung; the so-called cardiac lobe and dorsal lobe are examples.

The *cardiac lobe* is formed when a fissure occurs between the cardiac segment

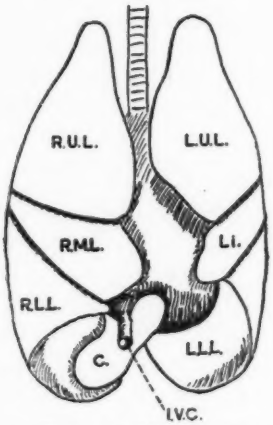

Fig. 1.—Thoracic Viscera of a Cat.

R.U.L., right upper lobe; R.M.L., right middle lobe; R.L.L., right lower lobe; C, cardiac lobe; L.U.L., left upper lobe; Li., lingula; L.L.L., left lower lobe; I.V.C., inferior vena cava.

of the right lower lobe and the remainder of this lobe; the name " lower azygos lobe " has also been given to this abnormality, but it is misleading and should be discontinued. Allen (1881) and Dévé (1900) noted that this lobe is an example of atavism, since the cardiac segment in man corresponds to a well-formed lobe in the lower mammals (Fig. 1). Dévé found traces of a cardiac fissure in 35 per cent. of human right lungs. The cardiac segment is supplied by a separate branch of the right lower bronchus, having its orifice on the medial wall of the bronchus between the orifices of the dorsal and anterior basic bronchi. In the left lung the cardiac segment is incorporated in the anterior basic segment and its bronchus is a branch of the anterior basic bronchus. A partial fissure outlining this area is occasionally seen in the left lung; Dévé mentions two examples of the left cardiac bronchus having a separate origin from the lower bronchus. We have seen one (Fig. 21, Plate XII). Keith (1933) suggests that the disappearance of the cardiac lobe in the higher

mammals is due to the development of the upright posture, with consequent apposition of the heart and diaphragm.

The *dorsal lobe* is formed by a fissure which partly separates the dorsal segment of the lower lobe from the remainder of the lobe. In 180 cases Dévé (1900) observed this fissure 40 times on the right side and 14 times on the left; the condition was bilateral in 12 cases. Lucien and Weber (1934) gave a full account of the bronchial supply of the dorsal lobe, and Churchill and Belsey (1939) discussed the surgical resection of this area, the independence of which had been stressed by Nelson (1934).

EXAMPLE 1 (Fig. 2).—Autopsy specimen from a female child, aged 1 year, who died of influenzal bronchopneumonia. In the right lung there was a fissure demarcating the anterior part of the cardiac segment and another outlining the lateral part of the dorsal segment. (See also Fig. 10, Plate IV.)

Abnormalities of lobation due to additional or deficient fissures are common in other mammals; Chase (1942) observed them in 86 out of a series of 458

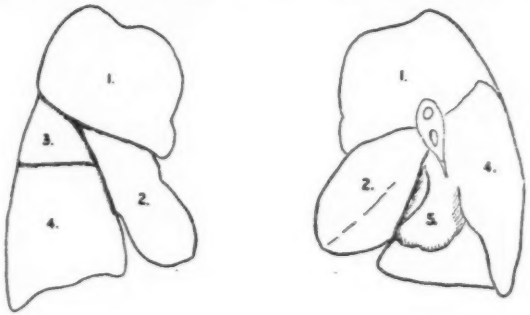

FIG. 2 (EXAMPLE 1).—RIGHT LUNG OF A CHILD.
A, Lateral view; B, medial view. 1, Upper lobe; 2, middle lobe; 3, dorsal lobe; 4, remainder of lower lobe; 5, cardiac lobe.

Rhesus monkeys, and Fig. 1 shows the lungs of a cat in which a complete fissure isolated the lingula of the left upper lobe.

Anomalies of lobation are readily understood when it is realised that additional fissures always occur between broncho-pulmonary units. Early in its development the surface of the lung somewhat resembles that of a blackberry, having a number of small protuberances each of which consists of a mass of mesoderm enclosing a growing bronchial bud. It is easy to imagine clefts developing between these growing areas, and thus, in the fully developed lung, any broncho-pulmonary segment or sub-segment may be separated by a fissure from its neighbours. To name such a sequestered area a "supernumerary lobe" is strictly correct, since it is an addition to the average number of lobes, but confusing because the term implies that extra lung tissue is present. In fact, the internal structure of the lung is normal and the abnormality is purely one of division; it is the fissure which is supernumerary.

As long ago as 1885 Maylard regarded additional fissure formation as the commonest pulmonary abnormality. Many examples have been recorded

since then, but their true nature has often been unrecognised and they have been regarded as additional lobes. Thus the abnormal lobes described by Geddes (1910) were clearly due to additional fissures separating some of the apical sub-segments of the right upper lobe, and the "upper azygos lobe, not associated with an abnormality of the vein" described by Stibbe (1919), is also an example of an intersegmental fissure in the right upper lobe.

Additional fissures may be visible in radiographs under normal conditions; associated pleurisy may render them very obvious (Fig. 6, Plate II). Two examples which follow illustrate additional fissures discovered at routine autopsy examinations.

EXAMPLE 2 (Fig. 3).—Autopsy specimen from a woman aged 39; accidental death by electrocution. The lungs had the following additional fissures: (1) between the two segments of the right middle lobe; (2) between the anterolateral and apicoposterior segments of the left upper lobe; (3) between the lingula and the remainder of the left upper lobe.

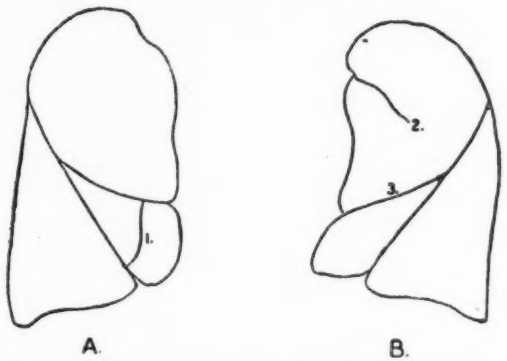

FIG. 3 (EXAMPLE 2).—LUNGS OF A WOMAN AGED 39.
A, Right lung, lateral view; B, left lung, lateral view. (For explanation see text.)

EXAMPLE 3 (Fig. 4).—Autopsy specimen from a man, aged 64, who died of cardiac failure. The lungs had the following additional fissures: *Right upper lobe :* (1) between the anterolateral and apical segments; (2) between the anterior and posterior apical sub-segments; (3) between the apical and posterolateral segments; (4) between the anterolateral and posterolateral segments. *Right middle lobe :* (5) between the anterior and lateral middle segments; (6) between two subdivisions of the anterior middle segment. *Left upper lobe :* (1) between the anterolateral and apicoposterior segments; (2) between the lingula and the remainder of the lobe; (3) between the anterior and lateral segments of the lingula. *Left lower lobe :* (4) between the posterior and middle basic segments; (5) between the anterior and middle basic segments; (6) between the dorsal and posterior basic segments.

(*b*) *Deficient Fissures*

The normal pulmonary fissures are often incomplete and sometimes absent. In a study of 277 lungs, Kent and Blades (1942) found that the lesser fissure of the right lung was absent in 21 per cent. and markedly incomplete in 67

per cent.; the main fissure was incomplete in 30 per cent. of both right and left lungs. Absence of a fissure has often been mistaken for absence of a lobe.

(c) Fissures due to Displaced Structures

(1) *Lobe of the Azygos Vein.*—The anatomy and radiology of this abnormality have been discussed many times. It was first noted by Wrisberg in 1777 and is sometimes called Wrisberg's lobe. Early writers thought that this lobe was a true supernumerary structure, but as far back as 1870 Cleland suggested that it was formed by a partial splitting of the normal right upper lobe by an abnormally placed azygos vein. In 1915 Holtby pointed out that the posterior cardinal vein in human embryos from 15 to 20 mm. long occupies a well-defined pleural fold outside the upper part of the lung, so that it would be easy for the apex of the lung to grow up on either side of the vein. When this

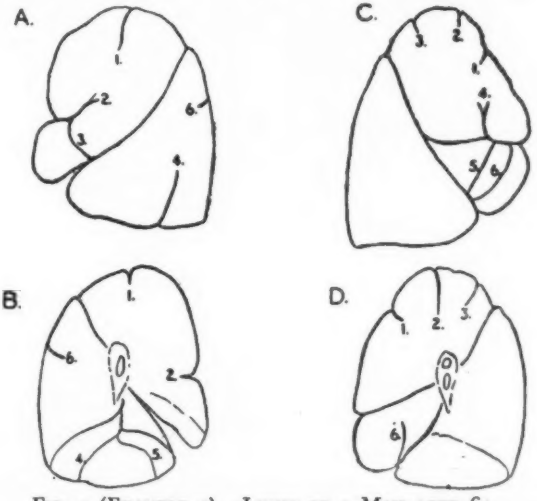

FIG. 4 (EXAMPLE 3).—LUNGS OF A MAN AGED 64.
A, Left lung, lateral view; B, left lung, medial view; C, right lung, lateral view; D, right lung, medial view. (For explanation see text.)

happens part of the right upper lobe is separated from the remainder by a mesentery-like fold of pleura enclosing the azygos vein, giving the characteristic radiological shadow (Fig. 7, Plate III). Mass radiographic studies suggest that the lobe of the azygos vein occurs in about 0·1 per cent. of human subjects (Clive, 1943); it appears to be normal in the porpoise (Allen, 1881; Cairney, 1923).

A simple method of showing that the lobe of the azygos vein is a part of the normal right upper lobe is to demonstrate its bronchial and blood supply, but in very few cases have even the bronchi been investigated. The fullest description is that by Cairney (1923), who dissected and made a model of the branches of the eparterial bronchus in a right lung which had a lobe of the azygos vein. He found that the apical branch had three divisions: anterior, which supplied the abnormal lobe; posterior and lateral, which supplied the

remainder of the apical segment of the right upper lobe. Gruber (1870), Dévé (1899) and Geddes (1910) all described the "medial apical" branch of the eparterial bronchus supplying the lobe of the azygos vein, and in each case the bronchus was kinked downwards under the vein to reach the lobe. Nelson and Simon (1931) recorded a similar finding, and it was also seen in a personal case (Example 4, Fig. 5).

As the lobe of the azygos vein is formed fortuitously during development by the failure of the vein to slide medially over the growing lung apex, one would expect this lobe to vary in size and to include a variable amount of the bronchial system of the right upper lobe. Stibbe (1919) stated that the fissure of the azygos vein might cut the lobe in any position from horizontal to vertical. This is well illustrated by the cases described below (Examples 4 and 5). The apical branch of the eparterial bronchus usually has three branches, two vertical (anterior and posterior) and one lateral. In Example 4 the azygos

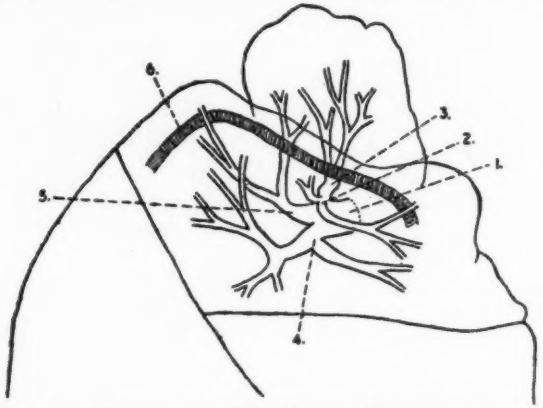

FIG. 5 (EXAMPLE 4).—TRACING OF A POST-MORTEM BRONCHOGRAM OF RIGHT UPPER LOBE, ANTEROLATERAL VIEW.

1, Eparterial bronchus; 2, lateral branch of apical bronchus; 3, remainder of apical bronchus (note the medial kink beneath azygos vein); 4, anterolateral bronchus; 5, posterolateral bronchus; 6, azygos vein.

vein divided both the apical and posterolateral segments of the right upper lobe; in Example 5 it chanced on the area between the vertical and lateral branches of the apical bronchus; while in Cairney's case only the territory supplied by the anterior vertical branch of the apical was cut off by the vein. Apart from a slight sketch by Dévé, the blood supply of azygos vein lobes has not previously been described. It was investigated in Examples 4 and 5 and the normal vascular pattern of the right upper lobe (Appleton, 1945; Boyden, 1945) was found to be present in both instances. All these findings confirm that the lobe of the azygos vein is merely a part of the normal right upper lobe and not an additional structure.

EXAMPLE 4.—A man, aged 23, who died of acute bronchopneumonic tuberculosis of the right lung. The lobe of the azygos vein was very obvious in the radiographs during life because of a tuberculous pleurisy with effusion extending into the abnormal fissure (Fig. 8, Plate III). The right lung was con-

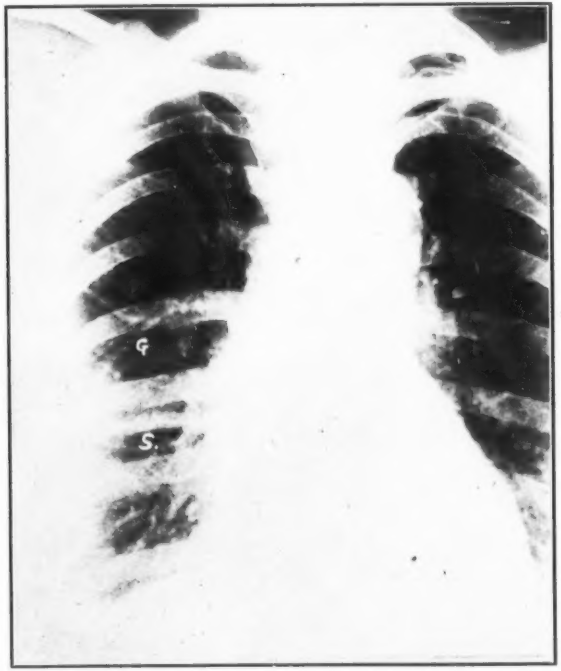

FIG. 6A. FIG. 6B.

FIG. 6.—SUB-DORSAL FISSURE OF THE RIGHT LUNG OUTLINED BY PLEURISY.
A, Postero-anterior radiograph; B, lateral radiograph; G, greater fissure; S sub-dorsal fissure.

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[*See page* 114.

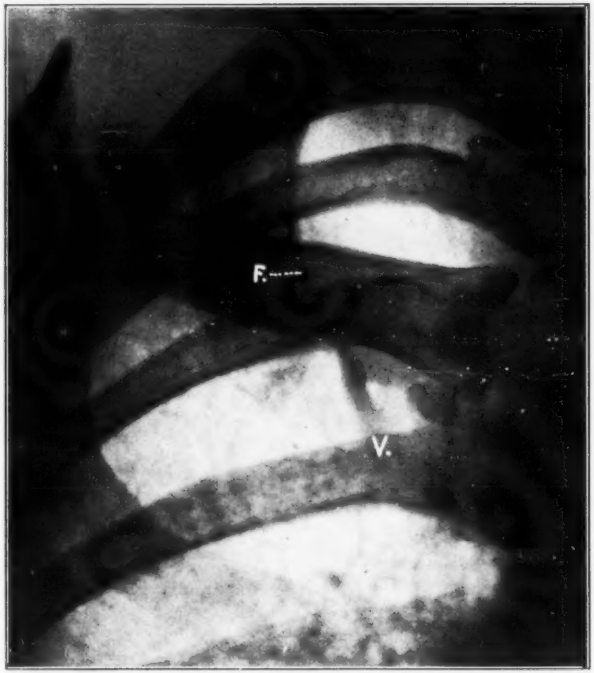

FIG. 7.—LOBE OF THE AZYGOS VEIN—POSTERO-ANTERIOR RADIOGRAPH.

F, Pleural fold; V, azygos vein. The loop of the vein is well shown.

FIG. 8 (EXAMPLE 4).—POSTERO-ANTERIOR RADIOGRAPH SHOWING RIGHT BASAL CONSOLIDATION WITH PLEURAL EFFUSION AND LOBE OF THE AZYGOS VEIN.

To face Plate IV.
[*See pages* 115 *and* 116.

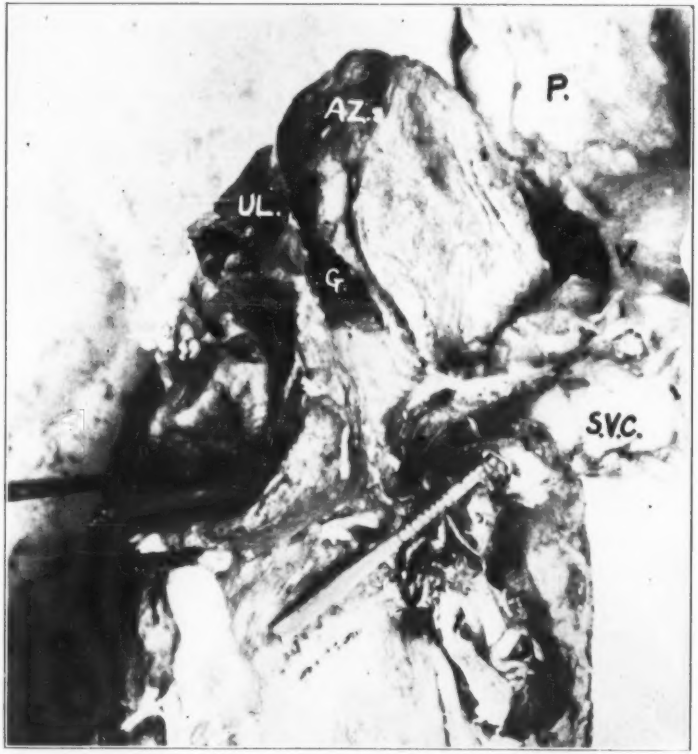

FIG. 9 (EXAMPLE 4).—RIGHT LUNG, ANTERIOR VIEW.

AZ, Lobe of the azygos vein; UL, apex of non-azygos portion of upper lobe; G, groove for azygos vein; P, parietal pleura; V, azygos vein; SVC, superior vena cava.

FIG. 10 (EXAMPLE 4).—RIGHT LUNG, MEDIAL VIEW.

AZ, Lobe of the azygos vein; UL, non-azygos portion of upper lobe; E, orifice of eparterial bronchus; C, cardiac lobe; P, parietal pleura; V, azygos vein; SVC, superior vena cava.

[To face Plate III.
[See page 117.

PLATE V.

FIG. 11 (EXAMPLE 6).—POSTERO-ANTERIOR BRONCHOGRAM (JANUARY, 1936).

F, Pleural fold enclosing azygos vein; E, eparterial bronchus; A, apical branch of eparterial bronchus.

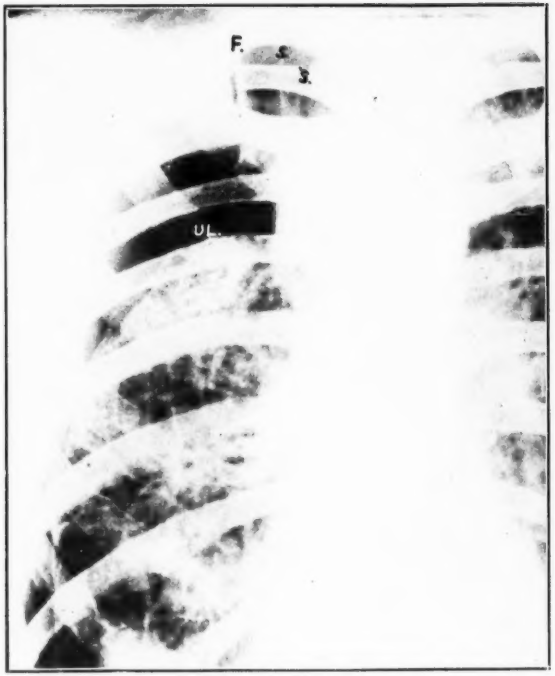

FIG. 12 (EXAMPLE 6).—POSTERO-ANTERIOR RADIOGRAPH (JANUARY, 1944) SHOWING TUBERCULOSIS OF RIGHT LUNG AND RIGHT ARTIFICIAL PNEUMOTHORAX.

F, Pleural fold enclosing azygos vein; S, adhesions; UL, non-azygos portion of upper lobe.

[To face Plate VI.
[See page 110.

PLATE VI.

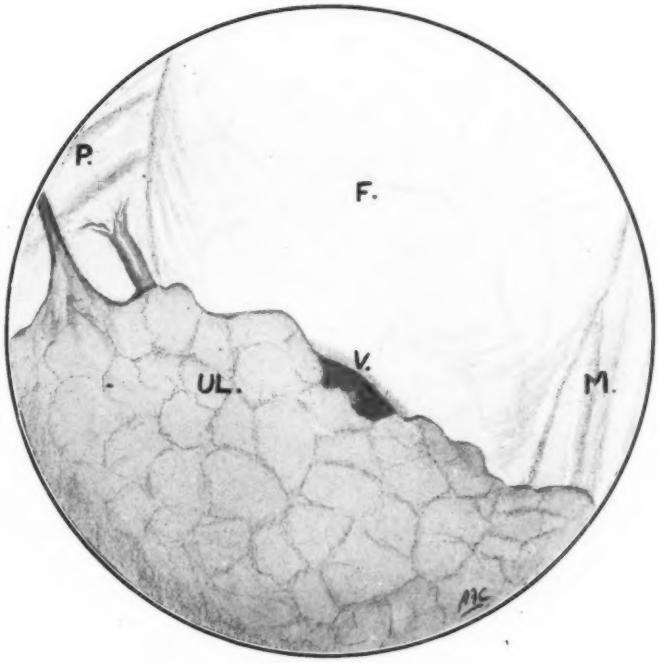

FIG. 13 (EXAMPLE 6).—THORACOSCOPIC APPEARANCE.

F, Fold of pleura enclosing azygos vein; V, azygos vein; M, anterior mediastinum; P, posterior chest wall; UL, apex of non-azygos portion of right upper lobe.

[*To face Plate V.*
[*See page* 119.

FIG. 14A.

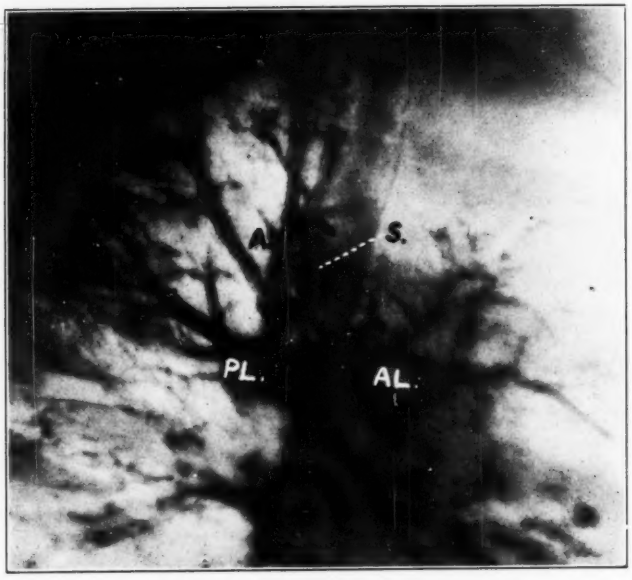

FIG. 14B.

FIG. 14 (EXAMPLE 7).—BRONCHOGRAM OF RIGHT UPPER LOBE.

A, Postero-anterior view; B, lateral view; E, eparterial bronchus; S, bronchiectatic supernumerary bronchus; A, apical bronchus; AL, anterolateral bronchus; PL, posterolateral bronchus.

[*To face Plate VIII.*
[*See page* 121.

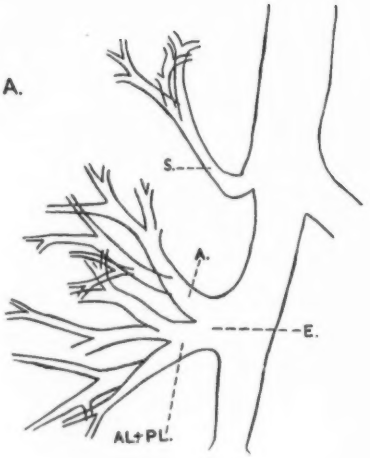

FIG. 15A.

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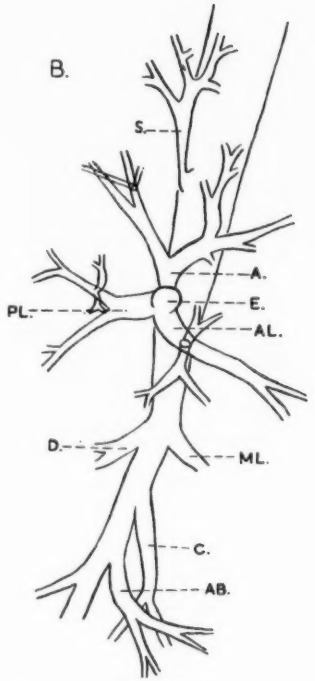


FIG. 15B.

FIG. 15A (PLATE VIII) AND FIG. 15B (EXAMPLE 8).—RIGHT BRONCHOGRAM.

A, Postero-anterior view; B, lateral view; E, eparterial bronchus; S, supernumerary bronchus; A, apical bronchus; AL, anterolateral bronchus; PL, posterolateral bronchus; ML, middle lobe bronchus; D, dorsal bronchus; C, cardiac bronchus; AB, anterior basic bronchus (hypoplastic).

[To face page 117.
[See page 121.

solidated and covered with a fibrinous exudate which had to be stripped off (Fig. 9, Plate IV); this also revealed a small fissure demarcating the anterior margin of the cardiac segment (Fig. 10, Plate IV). No other congenital abnormalities were found. The distribution of the bronchi was found by radiographs, after they had been injected with iodised oil, and then both bronchi and vessels were dissected to show their mode of branching. This method gives an excellent demonstration of the bronchial distribution (Fig. 5) and, by reducing the amount of dissection, enables the specimen to be preserved almost intact for museum exhibition. The anterolateral branch of the eparterial bronchus was unusually large, and its lateral division arose almost immediately, thus giving the appearance of a fourth branch of the eparterial bronchus. This variation is not uncommon (Foster-Carter, 1942—" Type 5 "). The apical bronchus had a lateral branch which supplied part of the non-azygos portion of the lobe, and the main stem of the apical was then kinked medially under the azygos vein before turning upwards into the

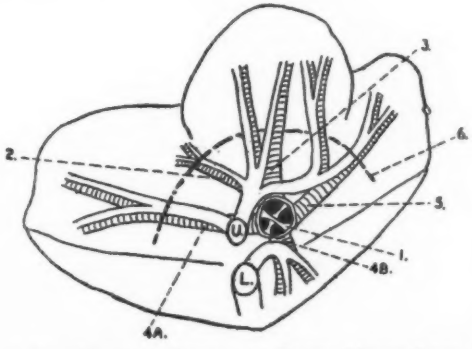


FIG. 16 (EXAMPLE 4).—ARTERIAL SUPPLY OF RIGHT UPPER LOBE, MEDIAL VIEW; ARTERIES PLAIN, BRONCHI SHADED.

U, Upper branch of pulmonary artery; L, lower branch of pulmonary artery; 1, eparterial bronchus; 2, lateral branch of apical bronchus; 3, remainder of apical bronchus; 4A, anterior branch of anterolateral bronchus; 4B, lateral branch of anterolateral bronchus; 5, posterolateral bronchus; 6, position of azygos vein.

abnormal lobe. The posterolateral bronchus had two branches, one of which supplied the apex of the non-azygos portion of the upper lobe while the other was directed upwards and medially into the lobe of the azygos vein. It will be seen that both the apical and posterolateral segments of this right upper lobe were divided by the vein. The arterial supply of this lobe is shown in Fig. 16. The upper branch of the pulmonary artery had two branches; one accompanied the anterior division of the anterolateral bronchus, while the other, curving backwards over the eparterial bronchus, gave branches which accompanied the divisions of the apical bronchus, and a " recurrent " branch (Appleton, 1945) which followed the posterolateral bronchus and its branches to the azygos vein lobe and the apex of the non-azygos portion. The lower division of the pulmonary artery gave an ascending branch to the upper lobe which accompanied the lateral branch of the anterolateral bronchus. The veins draining the lobe of the azygos vein joined branches from the remainder of the apical and posterolateral segments before draining into the superior pulmonary vein.

EXAMPLE 5.*—A man, aged 60, who died of a carcinoma of the left bronchus. The right lung had a lobe of the azygos vein, but was otherwise normal, and no other congenital abnormalities were found in the body.

The eparterial bronchus (Fig. 17) had three branches, but two of these, the apical and posterolateral, arose from it by a short common stem; this arrangement resembles that in the left lung and is not infrequently seen (Foster-Carter, 1942—" Type 2 "). The anterolateral branch was large and its lateral division supplied most of the lateral surface of the upper lobe. The posterolateral branch was correspondingly small. The apical bronchus first gave two lateral branches to the upper border of the non-azygos portion of the lobe, and its main trunk continued vertically upwards to supply the lobe of the azygos vein. Thus it will be seen that the vein, in this case, cut the apical segment into two parts.

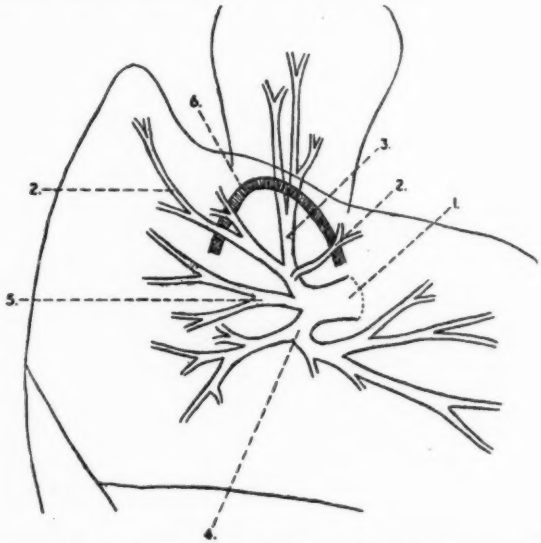


FIG. 17 (EXAMPLE 5).—TRACING OF POST-MORTEM BRONCHOGRAM OF RIGHT UPPER LOBE, ANTEROLATERAL VIEW.

1, Eparterial bronchus; 2, lateral branches of apical bronchus; 3, remainder of apical bronchus; 4, anterolateral bronchus; 5, posterolateral bronchus; 6, azygos vein.

The arterial supply of this right upper lobe is shown in Fig. 18. The upper branch of the pulmonary artery supplied the whole upper lobe; there were no ascending branches from the lower division of the artery. The upper artery had three branches: the first of these supplied the whole apical segment, including the lobe of the azygos vein, except for a small anterior lateral part, and gave a recurrent branch to the posterolateral segment; the second branch supplied the lateral part of the anterolateral segment and also the small lateral part of the apical segment mentioned above; the third branch supplied the anterior part of the anterolateral segment. The vein draining

* This case was reported briefly by Atkin (1934). The specimen was re-examined by the author in 1943, using the methods described above, to ascertain the bronchial and blood supply to the abnormal lobe. A note on the bronchi in this case was subsequently published by Brock (1944).

the lobe of the azygos vein joined branches from the other parts of the apical segment before draining into the superior pulmonary vein.

EXAMPLE 6.—A man of 22 was examined in January, 1936, when he complained of hæmoptysis. A bronchogram (Fig. 11, Plate V) showed a normal right bronchial tree and there was a lobe of the azygos vein (not recognised at this time) which appeared to be supplied by the apical division of the eparterial bronchus. The patient was not seen again until October, 1943, when he was found to have active pulmonary tuberculosis. A radiograph showed extensive infiltration with several cavities in the right lung. A right artificial pneumothorax was induced. In the subsequent radiograph (Fig. 12, Plate V) the fold of pleura enclosing the azygos vein was at first thought to be an adhesion. The true condition was recognised later, and the radiograph was then interpreted as showing the abnormal lobe held up by apical adhesions within the pleural fold of the azygos vein and well above the remainder of

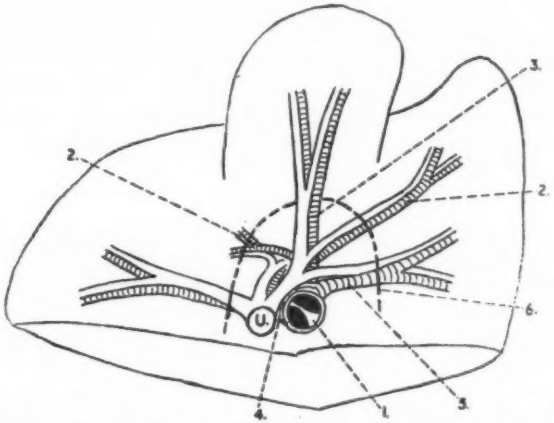


FIG. 18 (EXAMPLE 5).—ARTERIAL SUPPLY OF RIGHT UPPER LOBE, MEDIAL VIEW; ARTERIES PLAIN, BRONCHI SHADED.

U, Upper branch of pulmonary artery; 1, eparterial bronchus; 2, lateral branches of apical bronchus; 3, remainder of apical bronchus; 4, anterolateral bronchus; 5, posterolateral bronchus; 6, position of azygos vein.

the right upper lobe. This was confirmed at thoracoscopy (Fig. 13, Plate VI), the thin, translucent pleural fold extending from the anterior mediastinum to the paravertebral region and upwards to the apex of the pleural cavity. The azygos vein lay in the lower edge of this membrane, which appeared to inflate and deflate with respiration.

This example illustrates the practical value of recognising the presence of a lobe of the azygos vein in a tuberculous patient undergoing right-sided artificial pneumothorax treatment.

(2) *Sequestration of Lung due to an Abnormal Artery.*—Brown and Robbins (1944) described a congenital cystic condition of the posterior basic segment of the left lower lobe. The segment was separated from the rest of the lung by well-developed fissures and was supplied by an abnormal artery which arose from the thoracic aorta. Pryce (1946) has recorded a number of similar cases in which an anomalous artery was associated with functional isolation

of the part of the lung which it supplied. The bronchi in these sequestered areas were often dilated and cystic, and they did not communicate with the remainder of the bronchial tree.

(3) *Fissure for the Subclavian Artery.*—The only other example of a pulmonary fissure due to a displaced structure which we have found in the literature was recorded by Monod (1911). He described, briefly, a right lung having a bifurcation of the apex, 3 cm. deep, due to the subclavian artery.

II. Abnormalities of the Bronchi

(*a*) *Supernumerary Bronchi*

These are comparatively rare. Bremer (1932) described a 16·5 mm. human embryo having a large bronchus arising from the trachea on the right side. In a series of 80 human embryos he found tracheal buds in 3 others, and in 2 cases two buds were present on the same trachea. This relatively frequent finding of tracheal buds in the human embryo, compared with the very few reported cases of tracheal bronchi in the adult, suggests that many of these buds must be resorbed into the tracheal walls. Those which survive may become tracheal diverticulæ, accessory bronchi to the upper lobe, to an accessory apical lobe or to a rudimentary third lung in the mediastinum (Stibbe 1929, see below). If the stalk remains patent, the rudiment may be distended into a tracheocele. Stibbe (1929) described the case of a woman, aged 46, who had a right-sided aortic arch, above which was a large cyst-like body wedged behind the manubrium sterni. The mass was attached to the trachea 1 inch above the carina by a thread-like, tubular pedicle which was patent. Section of the tumour showed rudimentary bronchial and alveolar tissue resembling that found in accessory lungs. Chiari (1891) described cases of single pulmonary cysts on the right side communicating with the trachea, and Schiedegger (1936) mentioned two examples of a tracheal accessory lobe.

Some accounts of so-called supernumerary bronchi are open to question. Unless there is clear evidence that all the normal bronchi are present, as well as the abnormal bronchus, it is always possible that the latter is merely a normal branch arising in an abnormal position rather than a true supernumerary. This type of case will be described in the next section under the heading "Displaced Bronchi." McEwen (1937) gave an account of a bronchus arising from the trachea on the right side above the carina and leading to a cystic area at the right apex. The author calls this a tracheal accessory lobe, but the diagnosis was made on the bronchographic findings and there was no evidence of a fissure isolating this area. The tracheal bronchus in this case may have been supernumerary, but the published bronchograms suggest that it was more probably the apical branch of the eparterial bronchus displaced to an abnormal position. Brock (1944) described a similar case, and here again the bronchogram suggests a displaced apical bronchus. Ferguson and Neuhauser (1944) record a comparable case. Brock also described a supernumerary bronchus to the apex of the lower lobe, but in the published bronchogram the dorsal bronchus appears to have only two branches, so that the abnormal bronchus may be the apical branch of the dorsal bronchus arising higher up the stem.

FIG. 19A.

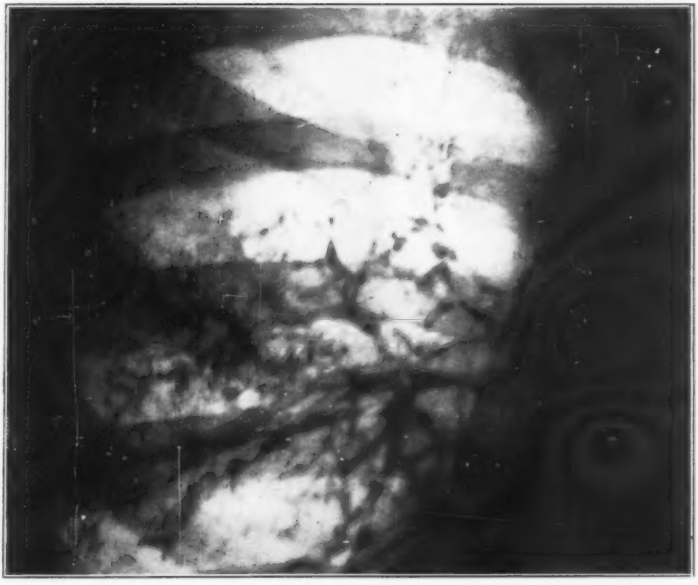


FIG. 19B.

FIG. 19 (EXAMPLE 9).—BRONCHOGRAM OF RIGHT UPPER LOBE SHOWING SPLIT EPARTERIAL BRONCHUS.

A, Postero-anterior view; B, lateral view.

[*To face page* 120.
[*See page* 121.

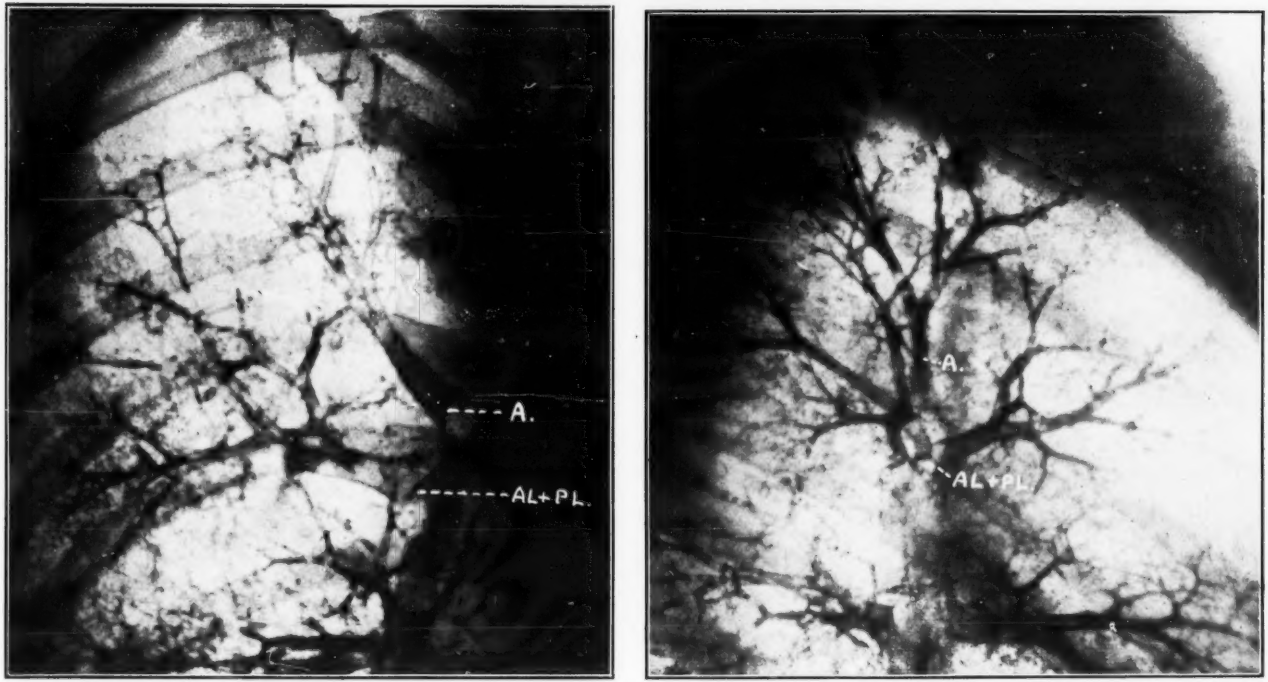


FIG. 20A. FIG. 20B.

FIG. 20 (EXAMPLE 10).—BRONCHOGRAM OF RIGHT UPPER LOBE SHOWING UPWARD DISPLACEMENT OF APICAL BRONCHUS. A, Postero-anterior view; B, lateral view. A, apical bronchus; AL + PL, common origin of anterolateral and posterolateral bronchi.

[To face Plate XII. [See page 122.

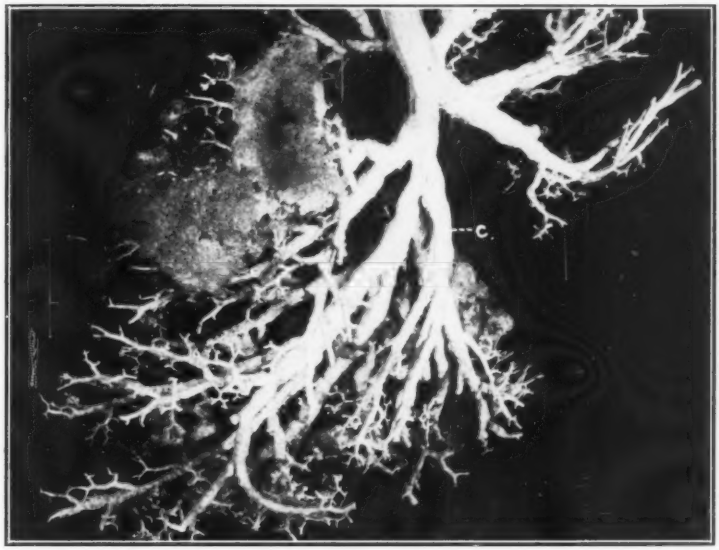


FIG. 21 (EXAMPLE 11).—CELLOIDIN CAST OF LEFT BRONCHIAL TREE, POSTEROMEDIAL VIEW, SHOWING SEPARATE CARDIAC BRONCHUS.
C, Cardiac bronchus.

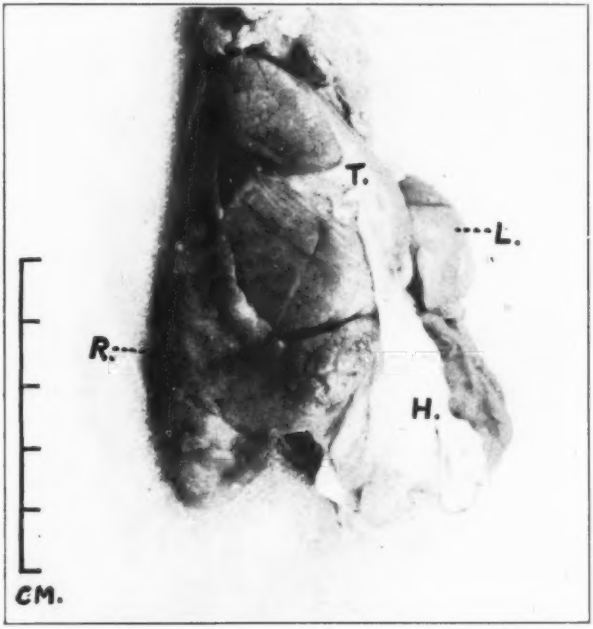


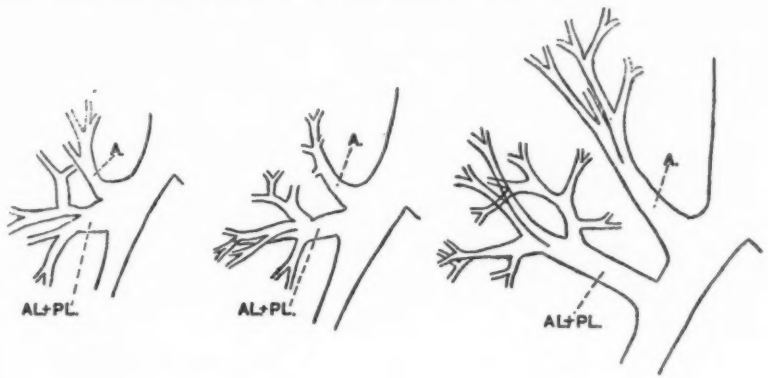
FIG. 22 (EXAMPLE 12).—THORACIC VISCERA OF AN INFANT, ANTERIOR VIEW, SHOWING HYPOPLASIA OF LEFT LUNG.
L, Left lung; R, right lung; H, heart; T, thymus.

[*To face Plate XI.*
[*See pages 122 and 123.*

The following are examples of true supernumerary bronchi discovered by bronchography:

EXAMPLE 7 (Fig. 14, Plate VII).—A bronchogram of a female child. A tiny bronchus arises from the trachea on the right side at the level of the carina and leads into a small cystic area. The lateral view shows that all three branches of the eparterial bronchus are present, as well as the supernumerary bronchus.

EXAMPLE 8 (Fig. 15, Plates VIII and IX).—A bronchogram of a young man. A large supernumerary bronchus arises from the trachea, 1 cm. above the carina, supplying the apex of the right lung. The eparterial bronchus, which arises slightly lower down the right bronchus than usual, is normal and all three branches are clearly visible. The right lower lobe appears smaller than normal and there is hypoplasia of the anterior basic bronchus.



A. B. C.

FIG. 23.—TRACINGS OF POSTERO-ANTERIOR BRONCHOGRAMS OF THE RIGHT UPPER LOBE SHOWING DEGREES OF SEPARATION OF THE APICAL BRANCH OF THE EPARTERIAL BRONCHUS.

A, Partial splitting of eparterial bronchus; B (Example 9), complete splitting of eparterial bronchus; C (Example 10); upward displacement of apical bronchus: A, apical bronchus; AL + PL, common origin of anterolateral and posterolateral bronchi.

(*b*) *Displaced Bronchi*

Displaced bronchial branches are relatively common; the most frequent example is an upward displacement of the apical branch of the eparterial bronchus. This may vary in degree from a partial splitting of the parent bronchus, which is quite common, to a frankly displaced apical branch arising from the trachea (Fig. 23). It is the latter abnormality which is sometimes mistaken for a supernumerary bronchus to the upper lobe, but a displaced apical branch can always be recognised by the fact that the " eparterial " bronchus, in such a case, has only two branches instead of three.

EXAMPLE 9 (Fig. 19, Plate X).—Split eparterial bronchus in a child. There is a deep cleft almost separating the apical branch of the eparterial bronchus from the common origin of the other two branches.

EXAMPLE 10 (Fig. 20, Plate XI).—Displaced apical bronchus. The "eparterial" bronchus has only two branches, anterolateral and posterolateral, the apical branch arising separately at the level of the carina, 2 cm. higher up. Compare this with a true supernumerary bronchus (Example 8, Fig. 15).

Instances of bronchial displacement in other regions of the lung have been recorded. The anterolateral branch of the left upper lobe sometimes arises from the lingula instead of the ascending branch (Churchill and Belsey, 1939; Brock, 1944), and Brock also recorded a case in which the anterolateral branch on the right side arose from the middle lobe bronchus. Displacement of the bronchi does not appear to be so common in the lower lobes, but Dévé (1900) mentioned two instances of a left cardiac bronchus, and we have seen one example.

EXAMPLE 11 (Fig. 21, Plate XII).—The left lung of a young woman was injected with celloidin via the bronchi, when investigating the broncho-pulmonary segments. The lung showed no external abnormality. After corrosion, the cast revealed a separate cardiac bronchus, arising from the medial aspect of the lower lobe bronchus between the origins of the dorsal and anterior basic branches, exactly as in the right lung.

(*c*) *Congenital Cystic Disease*

Cystic malformations of bronchial origin are not common; they are rarely found in routine post-mortem examinations of children. This is not the place for a discussion of cystic disease of the lung, nevertheless there is considerable evidence that cysts, with a preponderance of bronchial elements in their walls, do arise from congenital malformations of the developing bronchi. The subject has been reviewed recently by Willis and Almeyda (1943).

III. ABNORMALITIES OF THE LUNG PARENCHYMA

(*a*) *Accessory Lungs*

Isolated masses of rudimentary lung tissue are sometimes found both in the thorax and in the abdomen. These accessory lungs do not usually communicate with the bronchial tree, and they derive their blood supply from neighbouring vessels, often from the aorta. Histologically they consist of bronchial and alveolar elements resembling fœtal lung tissue. Accessory lungs are much more common on the left side than on the right and are often associated with congenital malformation or absence of the left diaphragm. Few infants with this defect survive, but in those that do, an intrathoracic accessory lung may be mistaken in a radiograph for a tumour or dermoid cyst. Rarely, there may be independent disease of the aberrant lung tissue (Freedlander and Gebauer, 1939). The exact origin of accessory lungs is obscure; the subject was reviewed by Cockayne and Gladstone (1919) and more recently by Davies and Gunz (1944).

(*b*) *Agenesis of the Lung*

This condition may involve the absence of a whole or part of a lung. Valle and Graham (1944) collected 38 proved cases from the literature and added 1 personal example. Three types have been described: true aplasia, in which the trachea leads into a single bronchus; cases in which there is a

small out-pocketing of the trachea representing the bronchus to the absent lung; and cases having a rudimentary bronchus leading into a small fleshy structure without lobes. The latter may be regarded as an extreme example of hypoplasia. Agenesis of the lung is twice as common on the left side as on the right. Ferguson and Neuhauser (1944) described 5 examples of agenesis which are of particular interest because in each case there was a congenital abnormality elsewhere in the body. These authors also record an instance of congenital absence of the right lower and middle lobes.

(*c*) *Hypoplasia of the Lung*

Comparison of various broncho-pulmonary segments in different lungs shows striking variations in the relative sizes of these units. Often one broncho-pulmonary segment will be large while the adjoining ones are correspondingly small. This is because each segment develops from a growing bronchial bud, and if one bud grows more strongly than its neighbours it tends to suppress them (see the anterior basic bronchus in Example 8, above). The same condition is sometimes seen in the pulmonary lobes. Marshall (1918) and Hepburn (1924) both described examples of right lungs in which the middle lobe was rudimentary and we have seen a similar case. Extreme hypoplasia of an entire lung has been mentioned in connection with agenesis, and occasionally a similar condition can be caused by the presence of abnormal structures in the chest which prevent the development of the lung.

EXAMPLE 12 (Fig. 22, Plate XII).—The thoracic viscera of a male infant who breathed after birth for a few minutes. The left half of the diaphragm was absent, and there was a pleural defect in the anterior mediastinal region; the left pleural cavity contained the stomach and most of the intestines; a loop of small intestine extended into the right pleural cavity through the anterior mediastinal defect. The right lung was expanded and appeared normal. The left main bronchus was very small and led into a tiny lung about the size of a hazel nut, which had a single shallow fissure. Bronchography revealed a branching bronchial system in the hypoplastic left lung; the right bronchial tree was normal. This case will be published in more detail elsewhere.

Summary

(1) Broncho-pulmonary abnormalities are being recognised with increasing frequency during life and have some clinical importance.

(2) A provisional classification of congenital defects of the lungs is outlined.

(3) The most common abnormalities are those of lobation; they may be due to additional or deficient fissures or to displaced structures such as the azygos vein.

(4) Abnormalities of the bronchi include supernumerary branches which may be distinguished from the more common displaced normal branches.

(5) Abnormalities of the parenchyma comprise true accessory lungs and varying degrees of agenesis and hypoplasia.

(6) Examples illustrating some of the more important abnormalities of the lung are described.

My thanks are due to Dr. G. E. Beaumont, Dr. J. L. Livingstone and Dr. Clifford Hoyle for permission to publish cases; to Dr. L. G. Blair for permission to reproduce some of the radiographs; and to Dr. Donald Teare for a supply of anatomical material.

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TROPICAL EOSINOPHILIA OR PULMONARY ACARIASIS

BY G. A. M. HALL

From the Cornish Riviera Sanatorium, Penzance

WRITING in 1943, Weingartner (1943) described as a new disease entity a condition characterised by distressing pulmonary symptoms, general ill-health and a high eosinophil count in the peripheral blood stream. Patients with this syndrome first came to his notice in Bombay in 1934. All his patients were seen in India, and though he found no relationship to a common mode of life, he formed the impression that it was commoner in persons who

resided near the sea. He discovered fortuitously that the symptoms responded readily to arsenic.

In 1940 Frimodt-Moller and Barton (1940) were able to select from a large number of sanatorium patients some 175 whose X-rays showed findings that they had come to describe as those of an " eosinophilic lung." All of these patients had more than 20 per cent. of eosinophilic cells in their differential blood counts. These authors do not seem to have used arsenic in the treatment of their patients.

In 1944, Carter, Wedd and D'Abrera (1944) searched the sputum of patients suffering from respiratory disorders for the presence of mites (acarina). These they were able to find in large or, more frequently, in small numbers in the sputum of seventeen of twenty-eight patients examined. The mites of ten different species of acarus were found, and all these species were common inhabiters of dusty atmospheres, especially those of storerooms and old houses. Three of their mite-infested patients showed high eosinophil counts, and after treatment with arsenic the number of mites found in the sputum was considerably reduced and many of the specimens found were degenerated.

Soysa and Jayawardena (1945), working in Ceylon, found that all of thirty cases of asthma with high eosinophil counts had been employed in dusty occupations, especially in ration store dumps. Mites were found in the sputum of eleven of the twenty-one patients whose sputum was examined for them. All their patients responded well to treatment with arsenic.

Apley and Grant (1944) report the case of an Englishman invalided from India with severe respiratory symptoms and a high eosinophil count. The eosinophils increased in number during a period of exacerbation of the pulmonary symptoms, but returned to normal spontaneously within a month. His exact occupation is not given. This patient appears to have had no treatment with arsenic at that time. It is to be assumed that the condition relapsed, as this patient is presumably the first of a series of five reported by the same authors in 1945 (Apley and Grant, 1945).

Lai (1945) gives a good description of the symptoms in his fifteen cases, stressing particularly the distressing nature of the cough, its occurrence with change of temperature, and the nocturnal paroxysms of dyspnœa.

Cases have been reported from parts of the world other than India which seem to conform with this syndrome. Thus Parsons and Smith (1944) reported an Englishman who after ten months spent in Cairo developed similar symptoms and findings and who responded to treatment with arsenic. Unfortunately, the exact occupation of this patient is not described.

From the Netherlands West Indies comes a description of eight cases by Van der Sar (1946). In each one mites were found in the sputum and photographs of the mite are reproduced. One case showed no eosinophilia. The occupations of the patients were varied, but contact with an infested environment is suggested by the occurrence of the condition in two sisters from the same building as had produced a case previously reported.

A case with an acute onset, developing eosinophilia somewhat late, but showing striking X-ray changes, was reported by Schleris in 1945. His patient, a serving soldier, had been on manœuvres in the Californian desert for several months before the onset of symptoms.

Clearkin (1944) reported a case from Georgetown in the Caribbean Islands in a man who was closely associated with horses but whose " asthma " did not clear when he avoided them.

Pulmonary nodules have been found in macacus monkeys (Davis, 1945), in which mites classified as " pneumonyssus " by Hoeppli were found. It is not known if an eosinophilia developed in these monkeys.

Case Reports

The purpose of this paper is to report two examples of severe respiratory symptoms with eosinophilia, as they show many points of interest.

Case 1.—A Squadron Leader, R.A.F., aged 39, was referred to me on December 31, 1945. Prior to enlistment he was employed in a bank and had never been abroad except for short holidays on the Continent. He spent the three years ending in April 1945 in India, chiefly in Bengal, though finally in Colombo. His service with the Air Force was in " Q " branch, and during the whole of his stay in India he had been employed in the supervision of stores of all kinds, both " dry goods " and equipment. During the few months preceding his return to England he had handled large quantities of crude paper in a dusty store in Colombo. His previous health had been good, except for a period in 1939 when he had suffered from dermatitis. He had never previously suffered from asthma or respiratory troubles of any kind, nor was there a history of allergic conditions in his family.

His present illness started with a cold in June 1945, two months after his return from India. He soon developed a severe and distressing cough, which occurred with any change of temperature, and especially when getting into bed and on first getting up in the morning. Exercise was liable to start paroxysms of cough. A scanty white and frothy sputum was raised from time to time, and on occasions this was seen to contain flecks of blood, thought by the patient to be due to the violence of his cough. There was no taste or odour to the sputum. His general health had definitely deteriorated. He had lost weight, felt ill and was short of breath. His great complaint was that he could " no longer keep up with his wife." He sweated easily and frequently felt faint.

He was overhauled and X-rayed in various R.A.F. and E.M.S. hospitals in August, October and November and was told that he was suffering from bronchitis. A final X-ray was reported as showing " increased markings at base of right lung, suggesting chronic bronchitis."

At his first consultation the cough was so troublesome as to be embarrassing. He looked ill, with a " china clay " white pallor and sweated easily. The chest expanded equally on the two sides. There were no abnormal percussion changes, but constant high-pitched squeaking râles were heard throughout both lung fields. The heart was normal but extra systoles occurred frequently. The blood pressure was 106/80. The spleen was not felt. An X-ray of the lungs showed the changes which are now recognised as those of the " eosinophilic lung "—namely, in addition to heavy trunks in the right basal area there was a very fine reticular mottling in the right basal and mid zones. The significance of these changes and the very characteristic symptoms were not appreciated at that time.

The patient was given a course of sulphathiazole and kept in bed. No improvement resulted and an exacerbation of the symptoms with fever developed at the end of January 1946. He was seen again on February 15, 1946.

Blood counts were made with the following results: Hæmoglobin, 96 per cent. Red blood cells, 5,480,000 per c.mm. Colour index, 0·88. White blood cells, 23,200 per c.mm. Mean diameter, 7·1 mu.

Differential Count.—Polymorphs, 7 per cent.=1,624. Lymphocytes, 5 per cent.=1,160. Monocytes, 20 per cent.=464. Eosinophils, 86 per cent.=19,952.

Examinations of the stools for ova and parasites gave negative results. Attention was then focused on the examination of such scant specimens of sputum as could be procured. On February 22 the pathologist reported: "Mucoid sputum with a little pus. At least 90 per cent. of the leucocytes are eosinophils. Mixed organisms present. Prolonged search did not reveal the presence of mites." On March 7 one degenerated mite was found, but the type was unrecognisable. Treatment with Carbarsone (Leucosone B.D.H.) was started on February 27, 0·2 gm. being taken night and morning for ten days. There was no exacerbation of symptoms; in fact, the patient started to improve on the fourth day. The increase in the number of eosinophil cells was, however, striking. On March 7 (fifth day of treatment) the total white count was 37,600, of which 90 per cent. were eosinophils; an absolute count of 33,840 eosinophil cells per cubic mm. of blood. On March 22 the absolute count of eosinophils was 3,348 (31 per cent. of 10,800).

By this time the patient was feeling well. He had no cough at any time. He was able to undertake normal activity and could once more "keep up with his wife." Since, however, the total eosinophil count on April 5 was still 2,496 (32 per cent. of 7,800), an intravenous injection of novarsenbillon 0·45 gm. was given on April 8. On April 17 a blood smear showed only 8 per cent. of eosinophils and the patient was returned to his unit.

Case 2.—A fruiterer of 38 years was seen in February 1946. His illness started soon after demobilisation from the Navy, in which he had spent five years in a mine-sweeper. His service had all been in home waters and he had never been to the tropics. Prior to his war service he had spent eighteen years in the same fruit store, and he returned to this work in August 1945. His work necessitated the handling of large quantities of vegetables and fruit, delivered to dusty store-rooms in jute sacks. Just prior to his illness he had spent hours of each day in the banana room, which was kept warm and moist.

His illness started in January 1946 and was at first thought to be an attack of influenza. He failed to recover and was referred to me on February 19, 1946. At that time he complained of cough and tightness in the chest. He went to bed feeling well and free from cough, but woke regularly at some time during the night with a choking sensation which forced him to cough. The cough was distressing but produced no sputum. He was never able to go to sleep again and spent the rest of the night out of bed. He sweated heavily at these times. On examination he looked ill, with a striking dead white pallor and an anxious facial expression. There was no cough and no dyspnœa at the time of the examination. There was some nasal obstruction. The lungs were completely free from abnormal physical signs. The heart was normal and the spleen was not felt. An X-ray taken on that day showed no abnormality. The blood count showed 8,000 white cells, of which 8 per cent. were eosinophils. On March 7 the patient was in no way better. He remained free from cough during the day, but was wakened in the early hours of the morning with an acute sense of distress, coughing and gasping for breath and unable to lie down again till after daylight. On this day his white count

was 22,200, of which 22 per cent. were eosinophils (4,884 eosinophil cells per cubic millimetre).

On March 14 treatment by carbarsone, 0·2 gm. night and morning, was started. The patient made steady improvement and returned to work early in April. On April 2 the eosinophils were 8 per cent. of a total count of 7,000 per c.mm. (560), and on April 30 were 6 per cent. of 8,000 (480).

Discussion

The presence of a high eosinophil count in conjunction with pulmonary symptoms has been known before Weingartner described his new disease entity. Engel (1935) described several cases of what he called " privet asthma " in which high eosinophil counts were noted. Loeffler's syndrome has reached the textbooks. The occurrence of some increase in the eosinophil cells in cases of bronchial asthma, especially those due to true allergy (Ratner, 1943), has long been known. Eosinophilia of any marked degree occurs otherwise only in infestation with parasites either dermal or alimentary.

The striking symptoms and the length of illness separate the condition under discussion somewhat clearly from Loeffler's syndrome, and as practically every assured case of eosinophilic bronchitis has been acquired in tropical countries it would seem wise to look for a specific etiological agent common to hot damp climates. The close association of all the patients described by Soysa and Jayawardena with dusty occupations, and the somewhat unexpected association of my two patients with similar conditions, naturally makes me favour the mite theory of Carter and his associates.

I am prepared to have the diagnosis in my second case disputed, as it was not confirmed by the finding of acari in the sputum. If, however, it is accepted it will be of value in showing that " tropical eosinophilia " may be contracted without residence in the tropics. It will suggest that " pulmonary acariasis " is a better name for the condition and that it must be considered in the differential diagnosis of chronic pulmonary diseases in patients who have never lived in the tropics. It will emphasise the importance of enquiring closely into the working conditions of all such patients.

Summary

The history of two patients suffering from distressing pulmonary symptoms and high eosinophil cell counts is given. One patient had never resided in the tropics. The working conditions of both patients prior to the development of their symptoms had included the passing of many hours in hot, damp and dusty store-rooms.

Arguments are evinced in favour of a mite infestation as the cause of the " new disease entity " of Weingartner, and it is suggested that the condition merits the name of " pulmonary acariasis."

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CASE REPORTS

BRONCHIAL CARCINOMA IN A CASE OF PULMONARY TUBERCULOSIS UNDERGOING ARTIFICIAL PNEUMOTHORAX TREATMENT

By DAVID FULTON and CHRISTOPHER ROLLESTON

THE simultaneous occurrence of pulmonary tuberculosis and carcinoma in the respiratory organs has been known for long, and the difficulties in diagnosis appreciated. The earliest reference that can be found is related by Norris and Landis (1924). Landis quotes from K. Wolff, who in the *Fortshitte der Medicin*, 1895, described 31 cases of malignant disease of the lung, in 13 of which tuberculosis also existed. Shaw (1901), however, accepted only 8 of these as being even possible and regarded most of them as doubtful. The only satisfactory proof is the finding of tubercle bacilli, and this was afforded some fifteen years later by Ross (1916), who had 2 cases in which tubercle bacilli were found in the sputum out of 60 cases of malignant disease. Ross adds that the frequency of healed tuberculous lesions found at autopsies is of no significance, as they are found with equal frequency in persons dying of other diseases. A similar opinion was given by Tudor Edwards (1942) that tuberculosis and cancer of the lung are occasionally associated, but there is no evidence of increased liability to carcinomata in tuberculous patients. This was the view also of Landis (1924), who wrote: "While there are some who believe the association of tuberculous and malignant disease is not infrequent, the majority of observers hold the view that the two diseases rarely co-exist. Among 662 autopsies at the Phipps Institute there has been no instance in which the two diseases occurred together." He had, however, seen 4 cases in which tuberculous lesions became quiescent, and the patients later died of malignant disease.

Lawrason Brown in 1925 wrote that Schwalbe reported 3 cases of cancer developing in the wall of a tuberculous cavity, and that Claude thought that tuberculosis is not uncommon in cancerous deposits. Lawrason Brown mentioned that one of his patients with pulmonary tuberculosis developed a primary carcinoma of the affected portion of the lung. Ormerod (1934) also reported a patient with active tuberculosis and carcinoma in the same lobe of the lung.

Case Report

C. B. was first seen by one of us (C. R.) on August 23, 1924, at the age of 28. His first wife had died from pulmonary tuberculosis that year. He had

enjoyed excellent health until he was gassed during the first Great War in 1916. Since then he had had a cough and a year prior to my seeing him a few specks of blood in his sputum. At the first examination his temperature was normal, his pulse rate 80 to 84, and there was no definite evidence of disease. But a month or two later diminished resonance was detected at both apices, and crepitations were heard at the right apex. Tubercle bacilli were then present in the sputum. He was admitted to Creaton Sanatorium two months later, and discharged after three months. By then he was free from cough, was not expectorating, and was able to resume work as a motor driver for the Post Office. Crepitations were still present at the right apex.

He remained in much the same condition for two years, when extension of disease in the right lung was noted, and crepitations were heard over the front of the left side of the chest. His weight began to diminish, and in five months' time had fallen from 9 stone 7 lbs. (when he left Creaton) to 9 stone. He was then put to bed, as his temperature was between 99° and 100°, and blood had reappeared in the sputum, which was found to contain tubercle bacilli again early in the next year. His general condition again improved, and the moist sounds in his chest diminished. He remained in much the same condition clinically, with crepitations audible at the inferior angle of the right scapula; but sputum became negative and his weight increased by a stone to 10 stone.

He remained well and in work till the end of 1933, when his weight had fallen to 8 stone 9 lbs. In the early part of the next year he had an attack of bronchitis with still further loss of weight. Two years later he was seen again after cough had been troublesome all the winter. Crepitations were heard from the apex of the right lung to the inferior angle of the right scapula. Numerous tubercle bacilli were found in the sputum. A radiograph showed some infiltration of the right apex, and some also of the left apex. He was kept in bed for two months, when phlegm had much diminished in amount, but physical signs remained the same. He was readmitted to Creaton and remained there for three months. He gained 12¼ lbs. in weight, and sputum became negative. Dullness and crepitations were still present at the right apex, but radiologically the shadows were much harder than on admission.

The morning after his return home he was taken ill with a fever between 99° and 100° and a productive cough with some hæmoptysis. There was cavernous breathing at the right apex, with râles extending to the inferior angle of the right scapula. He was admitted to hospital early in the next year and remained there for four months without much improvement, tubercle bacilli being present in the abundant sputum, while râles extended on the right to the inferior angle of the right scapula, and on the left to the middle of the vertebral border of the left scapula. The throat was very sore. Both vocal cords were very red, and there was some ulceration of the left vocal cord. The arytenoids and epiglottis appeared normal.

He was readmitted to Creaton Sanatorium two months later, where a right partial artificial pneumothorax was induced. The sputum was then small in amount and did not contain tubercle bacilli, nor were they ever found again. Over the whole of the right lung the breath sounds were well suppressed. Crepitations were heard on the left from the apex to the spine of the scapula. A radiograph early in the next year revealed partial collapse of the right lung. No adhesions were present. There was old disease of the left apex and upper zone. Weekly refills of 500 c.c. were continued for two months, when fortnightly refills were started and were continued till early in

the next year, when weekly refills were recommenced as the collapse was incomplete and an adhesion to the diaphragm was found. A mild pyrexia continued and his sputum was blood stained. The vocal cords were inflamed but showed no ulceration.

In the autumn of this year fortnightly instead of weekly refills were resumed. Nine months later his weight had dropped by a stone, and X-ray showed an adhesion at the lower border of the right lung, but otherwise a good collapse. No fresh infiltration was seen in the left lung. He was readmitted to Creaton Sanatorium for division of the adhesion, and on discharge had gained 5 lbs. in weight and had a negative sputum. X-ray examination showed an almost total collapse of the right lung with some adhesions in the apical and basal regions. The infiltration in the upper part of the left lung seemed more fibrous and was partly calcified.

Towards the end of the year fever returned and blood appeared again in the sputum. Radiologically the collapse of the right lung was satisfactory, with little change except for increased pleural thickening over the right apex; the upper mediastinal structures were somewhat displaced to the right, but the heart was slightly displaced to the left. The left lung had a moderate degree of disseminated tuberculosis, the nodules of which were partly calcified and partly suggestive of a recent active infiltration.

In view of the raised temperature, the repeated hæmoptysis, the physical signs, and the X-ray report it was decided to start a left-sided artificial pneumothorax. After a small initial fill, at which a high negative pressure was found, he became breathless from a complete spontaneous pneumothorax and had to have air removed twice on the same day.

Refills were discontinued. Pyrexia, 99° to 100°, continued. A month later breath sounds were heard all over the left lung. His condition deteriorated, he lost flesh rapidly, and complained of agonising pain in his rigidly held neck.

There was, however, little change in the condition of the lungs, and on June 4, 1943, an X-ray showed a right hydropneumothorax with partial collapse of the lung. The upper apical region showed fibrosis with displacement of the trachea to the right. There were some calcified foci in the right lung. No cavities were seen. An active infiltration was found in the left apical region. Healed bronchopneumonic foci were shown in the right upper lobe. No definite tuberculous lesion was found in the cervical spine. There was little change in his condition, the temperature still being raised from 99° to 101°, crepitations being heard at both apices and occasionally as far as the inferior angles of both scapulæ. He gradually became more querulous and difficult to manage, and the question of whether the tuberculous disease was spreading to the central nervous system was considered. He was admitted for further investigation to a War Pensions Hospital, but no tubercle bacilli were found in the sputum, nor was any evidence of tuberculosis of the spine or of the brain discovered. The blood sedimentation rate was high, 76 mm. in one hour and 119 mm. in two hours.

A month later breath sounds were suppressed throughout the right lung. He complained of generalised pains, and soon afterwards, in November 1943, he became incontinent of urine with plantar reflexes extensor for the first time. This, with the incontinence, again suggested tuberculous meningitis. He died suddenly on December 1, 1943, without a definite diagnosis being made.

At necropsy the larynx and trachea appeared normal, but the right main bronchus was completely obstructed about one inch from its origin. The

right lung was completely collapsed and the pleural cavity filled with blood. The pleura on this side was much thickened, especially over the diaphragm. The upper lobe was extensively infiltrated by a carcinoma which had arisen from the right main bronchus and was also infiltrating the tissues of the mediastinum and across into the left upper lobe. Several old tuberculous cavities with fibrosis were present in the right upper lobe.

Histologically the growth was composed of cuboidal or columnar cells growing in fairly large masses with very little gland tubule formation. In the section of parts of the upper lobe a mixture of tuberculous infiltration with typical giant-cell systems and carcinomatous infiltration was seen.

The left pleural cavity was obliterated by adhesions over the left apex and at the back of the left upper lobe, and also between the lower lobe and the diaphragm. There was carcinomatous infiltration from the mediastinum into the upper part of the upper lobe, and scattered through the rest of the lung there were numerous small white nodules varying from 0·5 to 2 mm. in diameter. Histologically these were deposits of carcinoma, but no tuberculous lesions were found in this lung.

Secondary deposits of carcinoma were present in the bodies of the 2nd, 3rd, 10th and 11th thoracic vertebræ, in the upper part of the sternum, and in the liver, portal glands and kidneys.

The skull appeared normal, but in the brain there was a well-marked right-sided hydrocephalus with a small secondary carcinomatous deposit lying just in front of the right foramen of Munro. Other secondaries were present in the body of the left caudate nucleus, the left optic thalamus, and two in the white matter just above the anterior horn of the left lateral ventricle. No evidence of tuberculous meningitis was found.

Histologically all the secondary deposits were similar to the primary growth in the bronchus.

Discussion

Failure to diagnose this case was due to the previous existence of well-marked tuberculous disease, and to the neurological symptoms which suggested meningitis. We put too little emphasis on the repeatedly negative sputum results in a patient with advancing illness, and so failed to have radiographs taken in positions other than the antero-posterior or to investigate the bronchial tree more thoroughly.

The diagnosis of these cases may be a matter of great difficulty not only because when the presence of the one condition is established the other is apt to be overlooked in explaining anomalous features, but also because they can be confused even if the possibility of a double diagnosis is admitted. Symptoms can be equivocal, and particularly if the upper lobe is affected, so can the physical signs. With a pneumothorax, as in this case, these difficulties are still greater. The radiological picture of the more usual types of growth, either atelectasis or a pulmonary or mediastinal tumour formation, is distinctive; but two of the less common appearances, secondary abscesses due to partial bronchial obstruction or to a necrosing growth and mottled infiltration due either to small foci of secondary septic change beyond a bronchial block or to diffuse lymphatic infiltration of the lobe with tumour cells, may well simulate tuberculous changes. The significance of a persistently negative sputum then becomes a paramount feature and clearly, from our experience

with this patient, calls for the help that bronchoscopy and perhaps branchography may give.

Summary

An account is given of a patient undergoing artificial pneumothorax treatment for old-standing fibrocavernous tuberculosis of the right lung who died from a bronchial carcinoma arising in the right main bronchus.

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A CASE OF SPONTANEOUS PERFORATION OF THE ŒSOPHAGUS

BY K. D. FOGGITT

From St. Hillier Hospital, Carshalton

SPONTANEOUS perforation of the œsophagus has recently been the subject of a detailed review by N. R. Barrett (1946). The following example presents a few points of particular interest.

Case Record

A married woman, aged 55, was admitted to hospital at 6 a.m. on March 28 this year with a diagnosis of perforated peptic ulcer. She complained that for the past six months she had experienced a pain in her back which she had noticed chiefly at night. On the night of March 27 she had vomited some blood and afterwards had been seized with an acute pain across the epigastrium and the lower dorsal region of her back. The pain had persisted, but she had had no further vomiting. There was no pain in the shoulders.

The patient was a well-nourished woman obviously in great pain, with a temperature of 98·2°. On admission, the extremities were cold and cyanosed. The pulse rate was 100, the blood pressure 180/100. The tongue was moist. The abdomen was rigid but not distended, and scarcely moved with respiration. Tenderness was only present on pressing forward the lower ribs. There was no loss of liver dullness. There were no abnormal physical signs in the chest, cardiovascular system or C.N.S.

In view of the cyanosis and character of the pain a tentative diagnosis of acute pancreatitis was made. But a few hours later some surgical emphysema appeared over the right clavicle, and as examination of the chest still revealed no abnormal signs, a perforation of the œsophagus was considered likely. A chest X-ray was requested to demonstrate the level of the perforation; barium was given, and the plates clearly showed an anterior perforation at the lower end of the œsophagus.

Twelve hours after admission the abdomen was opened through a left paramedian incision. The left lobe of the liver was rotated and the œsophagus mobilised and drawn down, revealing a longitudinal rent about 1 inch long

some 2 inches above the cardia. No obvious ætiology was found, and the rent was repaired with two layers of continuous catgut sutures. A rubber tube was inserted into the mediastinum anterior to the œsophagus and brought out through a stab incision in the abdominal wall. A gastrostomy was also done and the tube brought out through a separate stab incision. The abdomen was then closed in layers.

After the operation feeding was done entirely through the gastrostomy tube. Penicillin was given intramuscularly in doses of 20,000 units three-hourly, as well as locally into the mediastinal tube in 500 unit doses four-hourly. In spite of this, the patient gradually became more cyanosed, dyspnœic and distressed, and died two days after admission.

At necropsy a small shallow ulcer was found in the mucosa of the œsophagus just above the cardia. The perforation, with its sutures intact, was quite separate from this and at a slightly higher level. The mediastinum contained barium and necrotic material, but the perforation had not involved the pleural cavities, which were empty. Histological sections of the ulcer showed that it was in gastric mucosa, which extended to a higher level than normal. Section of the perforation revealed no sign of ulceration, but the outer surface of the œsophagus showed some necrosis which had been caused by the mediastinitis.

Comment

It is safe to assume that the ulcer was the cause of the pain in the back which the patient had experienced for the past six months, and that some bleeding from it had initiated the vomiting. This, associated with an element of spasm, was responsible for the perforation.

Perforation into the pleural cavities was not a feature of this case, as it has been in most of the cases previously reported. This supports the contention that it was a truly spontaneous rupture; for if there had been some previous ulceration at the point of perforation, one would expect the pleura to have become adherent and so facilitate a burst into the pleural cavities.

My thanks are due to Mr. Kidd, F.R.C.S.Ed., who was in charge of this case, for permission to make this report, and for his advice and encouragement.

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REVIEWS OF BOOKS

Chest Surgery for Nurses. By J. LEIGH COLLIS, B.Sc., and L. E. MABBIT, S.R.N. Second Edition. Pp. 169. 116 Illus., 14 Plates. Baillière, Tindall and Cox. London. Price 7s. 6d.

The second edition of this excellent book appearing within two years of the first is a good indication of the demand for a book of this nature. The new edition is some 40 pages longer and contains new sections on breathing exercises, thoracic anæsthesia, the mechanism of coughing, electrocardiography and thoracoscopy. The chapters on breathing exercises and anæsthesia are both comprehensive and full of practical details for the nurse. They form a most useful addition to the volume.

Postural drainage still receives rather scanty consideration and there seems to be no mention made of its valuable adjunct, percussion therapy,

which is so extensively used in the treatment of bronchiectasis and lung abscess. A detailed and practical consideration of postural drainage positions should be possessed by every nurse.

The line drawings appear a trifle amateurish and detract from the general tone of the volume. The illustrations of the instruments are on too small a scale for easy recognition, but one hopes that with the easing of paper restrictions these will be considerably enlarged. The X-ray supplement could be improved; the bronchograms are indifferent and some of the lesions depicted are rather obscure. But these are minor criticisms.

This is an excellent book which has improved considerably since its first appearance. It comprehensively covers the whole subject of thoracic surgery from the nurse's point of view in a clear, simple style. It should be possessed and studied by every nurse, whether general or chest trained. W. P. C.

Journal of the History of Medicine and Allied Sciences. New York. Annual subscription in England, $8.50.

It is pleasant, in these days of hurry, to find a new journal dealing with so leisured a study as the History of Medicine. Edited by George Roser and published by Henry Schomann of New York, this quarterly is attractively produced on paper such as we rarely see over here, and the illustrations, many of them title pages and frontispieces from sixteenth-century books, are most successfully reproduced. It is probably too much to expect a medical historian to be also a literary giant, but some more vigorous editorial pruning might have improved the loose construction of an article by Phyllis Allen on medical education in seventeenth-century England and spared us, elsewhere, the confusion of a sentence beginning, "Besides, however, we know that, for instance . . ." But it is natural for the contents of a journal to be of varied merit, and there are many fascinating articles, and best of all is the first part of a delightful essay by Singer on Vesalius and Galen. This journal promises to be a valuable addition to the cultural aspects of medicine. J. V. S.

Tuberculosis in the West Indies. By W. SANTON GILMOUR, M.B. 221 pages, 12 photographs, 2 maps, 38 tables. National Association for the Prevention of Tuberculosis. Price 7s. 6d.

This book is a series of reports by Dr. Santon Gilmour on tuberculosis in Trinidad and Tobago, Barbados, Windward Islands, Leeward Islands, British Guiana and the Bahamas. For each colony the general approach is similar. First, a study of the environmental, social and racial backgrounds on the widest possible basis, then a study of the available information regarding tuberculosis and of the existing facilities for diagnosis and treatment. Dr. Gilmour records his own field-work, mostly tuberculin-testing and assessment of clinical types of tuberculosis, with some mass radiography and post-mortem studies added for Trinidad. On this information Dr. Gilmour is able to base his excellent and detailed Recommendations.

The unsatisfactory environmental background is given the importance it deserves. The detailed description of small, over-crowded unventilated housing with little or no sanitation is supported by the evidence of some excellently reproduced photographs. Indeed, so well does the author bring out the unsatisfactory conditions of living, coupled with ignorance of elementary hygiene, that a recorded tuberculosis death-rate for Trinidad only some 50 per cent. above that of England and Wales appears surprising. Dr. Gilmour

is careful to point out that the figures are suspect, as many returns are made by persons without medical knowledge, and must be regarded as a low estimate. He quotes a suggestion by an American worker that the rate for British Guiana should be multiplied by five, but doubts whether the error is of this magnitude.

The performance of over 16,000 intradermal tuberculin tests in Trinidad was a considerable achievement. It is to be regretted that the method of presenting the results is such that there is relatively little information for the labour expended. From the table on pp. 37-39 giving results as percentages only, without absolute figures, it is impossible to assess the importance of the variations found between different age, sex and environmental groups. Further, it is not possible to obtain from the percentages given the incidence of reactors, or non-reactors, in sub-groups without a lengthy calculation. The results of tuberculin tests in other islands, given in appendices and based on smaller numbers, are in many ways more informative on account of clearer presentation. The general conclusion of a high incidence of reactors in town, with a much lower incidence in rural areas, appears justified. It is of interest that very few individuals with a high degree of sensitivity were found, in contrast to what has been found in some parts of S. Africa. (The ruling of the table at the top of page 126 is misleading—there appear to be two groups of negatives to 0·1 mg., one of which belongs to the 0·01 mg. section of the table.)

The author found little evidence of a "primitive" type of disease, and considers that primary infection occurring in adult life is not a major problem, except in immigrants from rural areas to towns. A large proportion of cases, 80 per cent., were "advanced" when first seen, cases presenting themselves late. Some resistance to the disease is apparent, more in the East Indians than in Africans. Mass radiography would help to find a larger proportion of early and treatable cases. Non-pulmonary disease is rare, as is infection from milk.

Except in British Guiana, there were at the time of the survey very few specialised facilities for the diagnosis and treatment of tuberculosis. Treatment beds were usually in a ward of a general hospital, insufficient in number and used mostly for advanced and dying cases. Dr. Gilmour has accordingly an almost clear field for his Recommendations, which are excellent in their scope and detail. He very wisely emphasises the need for improving environmental conditions, especially housing. He builds up a plan for a dispensary system, with tuberculosis officers and health visitors, linked to sanatoria with full facilities for treatment. He recognises the difficulty of supplying such services to the more scattered communities and makes a good alternative suggestion that in these areas the work of the tuberculosis officer should be combined with that of medical specialist. The only criticism of these most important Recommendations is on the grounds of practicability. With the present difficulties of staffing our own sanatoria, it is questionable whether even five trained and experienced nurses to supervise one sanatorium in Trinidad will be available, quite apart from the need for health visitors and the staffing of sanatoria in other islands. Nor would everyone with experience of mass radiography agree that "the production of thousands of miniature films with great rapidity will be possible without much technical skill."

One or two minor comments on the layout of the book are called for. Some indication at the top of each page of the area concerned would be most helpful, as would a rearrangement of headings and sub-headings in the Index at the beginning. Especially, it should be made clear that pp. 1-87 and appendices A-E relate to Trinidad and Tobago, in the same way that each subsequent section is headed by the name of the island studied.

The real value of this report is in bringing together the available facts regarding tuberculosis in this area, in contrast to the lack of facilities to deal with the problem. Dr. Santon Gilmour had no need to perform a great deal of original fact-finding to obtain evidence of the size of the problem. He is able to formulate excellent Recommendations based on methods already well tried in this country. The N.A.P.T. are to be congratulated on organising this work, and it is to be hoped that further studies in other colonies will be forthcoming.

V. H. S.

Modern Treatment Year Book, 1946. Edited by SIR CECIL WAKELEY. *The Medical Press and Circular*, London. Price 15s.

This annual volume, as usual, embraces wide interests. Indeed, the number of subjects dealt with inevitably mars the articles of some contributors; so little space is available for the consideration of any one topic. The effort to cover so wide a field is a decided disadvantage, and added to this the articles are jumbled together in no discernible order.

Three articles touch directly on the thorax. Dr. Shafer contributes a detailed account of œsophageal hiatus hernia, its ætiology, symptomatology and treatment. This is outstanding and it is a pity that it is wedged away here and liable to be overlooked. Mr. Graham Boyce in a short article on abscess of the lung sensibly confines himself within his allotted space to a plea for the early transfer of cases of putrid lung abscess to the care of the thoracic surgeon. Primary atypical pneumonia is dealt with adequately by Dr. Wingfield.

APPEAL

THE EDITOR, BRITISH JOURNAL OF TUBERCULOSIS AND DISEASES OF THE CHEST.

August 30, 1946.

SIR,—

Reports and requests from UNRRA Missions in Europe make it abundantly clear that the greatest expressed need of doctors in the liberated countries is for medical literature covering the war years, so that they may bring themselves up to date with advances in unoccupied countries.

From time to time, appeals for medical literature for individual countries have appeared in the medical press, and the British Council, the Ministry of Information, the Royal Society of Medicine with its microfilm scheme and other bodies have done something to cope with the problem, but, as a whole, the needs of Europe have hardly begun to be met.

Even if UNRRA could spend its now scanty funds on medical re-education—which it is authorised to do only in the limited sense of informing doctors how to use drugs and other medical supplies supplied by UNRRA with which they may be unfamiliar—a special difficulty arises in the case of British medical and nursing literature on account of the paper shortage.

I am, therefore, appealing for complete sets of general and specialist medical and nursing journals covering approximately the war years. They should be sent to Dr. H. Hadaway, Room 1934, Health Division, UNRRA, 19, Portland Place, London, W.1 (Telephone: Langham 3090/341), and carriage will be repaid if requested. If it is felt that the sets cannot be given free of charge, a price should be stated before the sets are forwarded to us and we will try to obtain authority for their purchase. A particular request, which we have so far been unable to meet, is for a complete set of the *Lancet* (1939 to 1945 inclusive) for Hungary. I am, sir,

Yours faithfully,

NEVILLE M. GOODMAN,

Director of Health, European Regional Office, UNRRA.

MEETINGS OF SOCIETIES

JOINT TUBERCULOSIS COUNCIL

AT the last meeting of the Joint Tuberculosis Council tribute was paid to the late Dr. George Jessel, who was Vice-Chairman of the Council at the time of his death.

Dr. James Boyd, Chief Medical Officer, Ministry of Health, and Local Government of Northern Ireland, attended the meeting for the first time as observer on behalf of his Ministry, and was cordially welcomed.

The Council adopted a report prepared by the Committee on the subject of the re-definition of terms used in tuberculosis work. The report suggested the abolition of the present system of recording cases of tuberculosis, and suggested new classifications. New definitions of "quiescent," "arrested" and other terms were also suggested. The report was forwarded to the Minister of Health with a request that its recommendations be carried into effect. But the Council decided not to publish the report at the present time, as changes might be made after the Ministry had had an opportunity of considering the suggestions in detail.

Attention was drawn to the anomaly created by a recent ruling that where family allowances under the Government scheme are received by persons who are entitled to financial assistance under Memorandum 266/T, the 266/T allowances must be reduced by the amount of family allowance received, and the Council authorised a deputation to express to the Ministry the strong view that family allowances should have no effect upon allowances granted to tuberculous persons as such.

Arising out of resolutions adopted at a previous meeting, expressing alarm at the probable effect of the National Health Service Bill on the Tuberculosis Service, it was reported that a deputation representing the Joint Tuberculosis Council and the N.A.P.T. had met Sir Wilson Jameson. Sir Wilson had on that occasion said that there was no intention to detach preventive work from curative work. He had also given assurances on other points. The report of the interview was accepted without prejudice to any future action which the J.T.C. may consider necessary in relation to the Bill.

The question of bread rationing and its effects, if any, upon tuberculous persons in and out of sanatoria was referred to the Council's Committee on Nutrition for consideration.

Arising out of a resolution of the Tuberculosis Association, the Council appointed a special committee to consider the establishment of a Bureau of Tuberculosis Statistics. The new committee was empowered to explore the possibilities of collecting tuberculosis morbidity statistics, to plan statistical enquiries into selected problems, and to advise—or arrange for expert advice to be afforded—on the collection of tuberculosis data. The committee was made as representative as possible, its membership including a tuberculosis officer, a sanatorium superintendent, a thoracic surgeon, an orthopædic surgeon, a pediatrician, a radiologist, a bacteriologist and a medical officer of health. The Council decided to approach the Nuffield Foundation for assistance in the project.

The standardization of the forms of record used by local authorities in connection with their tuberculosis services was discussed, and a committee was asked to consider the present situation and, if possible, to suggest standard forms for universal use.

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